Tangeman (C. W.) Dr. B. Joy Jeffries.

Compliments of the author

KERATITIS BULLOSA
PERSISTENT HYALOID ARTERY
CHOLESTERINE IN THE EYE

BY

C. W. TANGEMAN, M.D.

ASSISTANT TO THE CHAIR OF OPHTHALMOLOGY

[Reprint "ARCHIVES OF OPHTHALMOLOGY," xvii., 1, 3, 4, 1888]

CLINICAL CONTRIBUTIONS FROM PROF. W. W. SEELY'S CLINIC, MEDICAL COLLEGE OF OHIO.

KERATITIS BULLOSA
PERSISTENT HYALOID ARTERY
CHOLESTERINE IN THE EYE

BY

C. W. TANGEMAN, M.D

ASSISTANT TO THE CHAIR OF OPHTHALMOLOGY

[Reprint "ARCHIVES OF OPHTHALMOLOGY," xvii., 1, 3, 4, 1888]

NEW YORK AND LONDON
G. P. PUTNAM'S SONS
The Knickerbocker Press
1889

KERATITIS BULLOSA.

By C. W. TANGEMAN, Cincinnati, Ohio.

ASSISTANT TO THE CHAIR OF OPHTHALMOLOGY AT THE MEDICAL COLLEGE OF OHIO.

THIS disease, though not of very frequent occurrence, has been described by a number of observers in the last century. Cases have been observed and reported from time to time, and the subject has received considerable attention, experimentally, at the hands of Prof. Leber, and yet the etiology of this disease is quite obscure. The scarcity of clinical material has made it difficult to study the affection with any degree of satisfaction. Experiments on the eyes of animals have yielded but few data, except to disprove the erroneous theory of Landesberg, who advanced the idea that the formation of vesicles on the cornea is purely mechanical, caused by a force from behind, as in increased intra-ocular pressure. It is now definitely fixed that no matter how great the intra-ocular pressure, the fluid in the anterior chamber does not cause œdema of the cornea, or is not permitted to pass through the membrane of "Descemet" unless the surface be broken. In a large number of the reported cases of "keratitis bullosa" there was neither increased intra-ocular pressure nor inflammation of the uveal tract recorded.[1] Saemisch makes an infiltration of the parenchyma of the cornea the first stage in this disease. He says that all the difference there is between parenchymatous infiltration of the cornea and keratitis bullosa is that in the latter, some time during the course of the disease, there is a formation of large vesicles, larger than phlyctenular vesicles, and not so round and regular in shape. Graefe,[1] when making a microscopic examination of the anterior wall of the described vesicle, finds that not only the epithelial layer, but also the anterior elastic membrane and some fibres from

[1] Graefe and Saemisch: *Handb. d. Ges. Augenhk.*
[2] Graefe: *Arch. Ophth.*, ii., 1, 206.

Reprinted from the Archives of Ophthalmology, Vol. xvii., No. 1, 1888.

the parenchymatous layer of the cornea, are separated to make up the covering of the vesicle. Possibly the smallest number of cases would show an involvement of the cornea to that depth. Usually, as Schweigger[1] states, he never *found* that more than the anterior epithelial layer was separated in this disease. Bullous keratitis must be differentiated from the various forms of vesicular keratitis or herpes cornea, which may occur without any previous inflammatory disturbance. It is safe to say that we have to deal with a separation of the superficial layer only in all of these various forms of inflammation; the contents are the same, but the condition of the cornea and the circumstances under which each form is developed are different. Warlemont, McKenzie, and others have described this disease as dropsy of the cornea,—a formation of vesicles or blebs, due to an escape of fluid from the anterior chamber through the corneal tissue. While this theory has been disproven, nothing satisfactory has been substituted. Since a few cases have been reported of keratitis bullosa in glaucomatous eyes the tendency has been to connect the etiology of the two diseases. Degenerative changes of the epithelial cells, causing an enlargement of the intercellular spaces, have most recently been advocated as the chief process that is going on in the corneal tissue, which looks so much like a parenchymatous inflammation of the cornea. Michel reports cases where he observed the formation of vesicles quite large on a cornea otherwise perfectly healthy.

The patient that came under the writer's observation at Prof. W. W. Seely's clinic is of special interest, since the vesicles appeared on both eyes simultaneously, and on an otherwise perfectly healthy cornea.

Mr. B., æt. thirty, laborer, white, robust, and healty, complained of a disturbance of his sight. Since the past week he had suffered acute attacks of pain, which seemed to get better one day and worse the next. A careful examination of his eyes showed three or four vesicles on each cornea, quite large, filled with a clear fluid. The patient said he had had previous attacks of a similar nature, and he had seen "blisters" form on the sight,

[1] Schweigger: Handbuch, 1873.

that would pass away again. The cornea showed distinct traces of an apparently superficial abrasion. Aside from these charges nothing abnormal could be found about the cornea. The chief complaint was an intense burning sensation and some redness of the ocular conjunctiva. The tension was normal, and the depth and contents of the anterior chamber looked natural. The patient was not suffering from any constitutional affection, but gave a distinct malarial history or tendency.

On the second day later, he presented himself again, and some new bullæ had formed. The patient was now put on large doses of quinine, with the effect of removing all symptoms of irritation, and preventing a recurrence of the vesicles on the cornea. The patient was kept under observation for some time, but there was not even a tendency to a return of the symptoms above recorded.

Here, now, we have a case of a rare disease, carefully watched, with an intermittent tendency, being absolutely cured upon the administration of an antiperiodic. Does this indicate any thing? Graefe, Schultze, Saemisch, and others have recorded cases with distinct periods of intermission, but fail to speak of the possibility of malaria having any etiological connection with this affection. In the above case all of the diseases usually given as causative of vesicular keratitis, viz., glaucoma, iritis, ichthyosis, inflammation of the uveal tract, degeneration of the corneal epithelium, corneal infiltration, and enlargement of the intercellular spaces, engorgement of the lymph spaces, increased intra-ocular pressure, or a mechanical force, were absent. Hansen some time since described a case of intermittent vesicular keratitis, but insists that it was caused by trauma to the corneal epithelium.

How these vesicles should occur on the cornea due to malaria is difficult to say, yet it would be no more impossible than when they follow vaso-motor disturbances (Michel), or when we have a serous effusion into the vitreous (Seely[1]), or, finally, in the case reported by Perlia,[2] where the formation of the vesicles was due to vaso-motor changes caused by migraine.

[1] Transactions of the Amer. Ophth. Soc., 1883.
[2] Zehender: *Monats. Bl.*, 1888.

PERSISTENT HYALOID ARTERY.

By C. W. TANGEMAN, M.D.,

ASSISTANT TO THE CHAIR OF OPHTHALMOLOGY AT THE MEDICAL COLLEGE OF OHIO.

THE function of the arteria capsularis, the manner of its development, and usual disappearance prior to birth, is of extreme interest to the physiologist. A careful study of the embryological development of the eye as we understand it to-day, accounts for many of the peculiar freaks that were formerly simply called congenital. Such cases as coloboma of the iris, choroid, optic nerve, or persistent hyaloid artery, are of interest to the oculist, and are just as thoroughly understood as the surgeon understands in what manner hare-lip or cleft palate are brought about.

No other organ of the human body has received such careful and persistent investigation; no other organ can offer to the anatomist such striking changes succeeding each other in so short a space of time as the eye. Just what day or hour the eye can first be recognized in the human embryo cannot be said, since it is so exceedingly difficult to obtain any number of specimens in condition for investigation at this early stage. It suffices to say in this connection that the eye is one of the earliest organs to appear in the embryo. The crystalline lens begins to form early from an involution of epiblast. It lies in the optic cup, completely isolated as we find it later, just in front of the space to be occupied in the vitreous. It appears to get its nutrition by absorption up to this time.

The walls of the cup grow rapidly all around except that portion which is opposite the middle of the cup. In the

Reprinted from the ARCHIVES OF OPHTHALMOLOGY, Vol. xvii., No. 3, 1888.

natural position of the eye this spot is on its under surface, and by not growing, there is a gap left, which is called the choroidal fissure. This fissure extends back into the optic stalk, and through its opening the mesoblastic structure gets into the interior of the eyeball, from which the capsule of the lens and the vitreous humor are formed as well as the blood-vessels. It is known to every one that the arteria centralis retina lies in the interior of the optic nerve, finding its way into the latter by the optic stalk closing around this branch of the ophthalmic artery during the early embryonic development. At an early stage the lens, as we described it above, is surrounded by a capsule which becomes vascular. The posterior surface of this capsule is supplied with blood from branches from the arteria hyaloidea, which gets into the interior of the eye in the same manner as is described above for the central artery of the retina. This small vessel (which is never accompanied by a vein) runs through the centre of the eyeball forward and as soon as it reaches the posterior surface of the lens it divides dichotomously. The small branches pass around the margin of the lens so as to gain its anterior surface, where they supply the anterior capsule and the membrana pupillaris with blood. In the human fœtus at birth these structures have disappeared; they seem to have been necessary for the development of the lens but are not requisite for its later maintenance. The membrana pupillaris disappears early, as a rule, by absorption, but occasionally cases are observed in which a portion of the membrana or of the hyaloid artery persists at birth and remains so during life. Cases have been reported where the pupillary membrana remains intact, but it is counted one of the greatest rarities to find this membrane to contain vessels. The hyaloid artery is seen sometimes in its entire length, but more frequently as a small shrivelled thread still adherent at its origin.

There is another class of cases where vessels carrying blood are found in the interior of the eyeball. Small branches are given off from the retinal vessels, but these formations are always found to be connected with some

pathological process. V. Ammon [1] gives a representation of this condition in his atlas. Recently more light has been thrown on this subject. Weber [2] produces small vessels carrying blood in the vitreous, experimentally, by a process of irritation. Schweigger [3] shows the presence of vessels in the eye as a pathological process. Coccius,[4] as early as 1859, reports an exceedingly interesting case where the vessels took origin from the retina; the vitreous remained sufficiently clear to permit a very satisfactory study of the whole process. It is certainly difficult to say how the vessel walls are formed, and how they make connection with the retinal vessels. While these cases are an anatomical curiosity and exceedingly rare, they must not be confounded with the condition above described, where a portion or all of the embryonic arrangement of vessels for the nutrition and development of the lens and the anterior portion of the eyeball fail to disappear at the proper time. Everbusch [5] observed a case where the embryonal remains seem to indicate that originally there are two systems of vessels in the vitreous, one reaching toward the retina and the other surrounding the lens. Everbusch suggests that many of the cases reported as persistent hyaloid artery are in reality only a haziness of the canal of Cloquet, which occurs on account of irritation. Czermak [6] presents a case where a small vessel leaves the optic papilla, runs directly forward through the vitreous to about its centre, and then returns, spirally winding around its own axis. This small vascular loop showed distinct pulsations, rising and sinking with each systole and diastole of the heart. It is of great importance that a correct diagnosis is made in these cases. Vassaux [7] gives an instance where an eyeball had to be enucleated on account of a persistent hyaloid artery, and when the eyeball was later examined it was found to contain a gliosarcoma which, in its growth, had drawn some

[1] "Krankheiten des Menschenauges."
[2] *Virch. Arch.*, vol. xvi., p. 410.
[3] ARCH. OF OPHTHAL., vol. ii.
[4] "Ueber Glaucom," etc., Leipzig, 1859.
[5] "Beitr. zur Embryologie," München, 1882.
[6] *Centralbl. f. prakt. Augenheilkunde.*
[7] "Persistance de l'artère hyaloïde," *Arch. d'ophthal.*, vol. iii., p. 502.

few blood-vessels forward, giving to them the appearance of persistent hyaloid artery.

In the case reported by Hirschberg[1] there can be scarcely any doubt but that the vessel formation as observed was due to a pathological process, since the patient was suffering with retinitis punctata in the other eye. The same condition is observed by Ulrich,[2] where, in a typical case of retinitis pigmentosa, the author finds thread-like opacities in the vitreous.

Unterharnscheidt[3] reports a case of persistent hyaloid artery where the remains of the vessel were still attached to the central artery of the retina and the capsule of the lens. The case was under observation for some time when finally the fine strand tore in two, due to an increasing myopia, leaving two small ends floating in the vitreous. W. S. Little[4] reported a very peculiar case where he discovered with the ophthalmoscope a vessel leaving the central artery, running forward into the vitreous, turning upon itself, and after reaching the fundus again dividing into three or four small branches, which are finally distributed over the retina.

The vessel had a red appearance, and seemed to carry arterial blood. The remainder of the fundus was normal. Vision was very much reduced, due to amblyopia from disuse, the patient being under treatment for convergent squint. L. later reported another similar case, but the artery here was attached to the posterior surface of the lens, contained no blood, and disturbed vision but little. (Of all the cases reported where blood was found in the vessel, it came from the region of the papilla.)

Schapringer observed[5] in a child a small artery leaving the papilla and extending into the vitreous, where, turning upon itself, it returned to the starting-point. A number of similar cases have been observed from time to time. In a case reported by Galezowski[6] there were two vessels, a vein

[1] *Centralbl. f. Augenheilkunde.*
[2] *Klin. Monatsbl. f. Augenheilk.*, vol. xxi., p. 140.
[3] *Klin. Monatsbl. f. Augenheilk.*, vol. xx., p. 449.
[4] "*Transactions Amer. Ophthal. Soc.*," 1881.
[5] ARCH. OF OPTH., vol. iii.
[6] *Recueil d'ophth.*, 1883.

and an artery. A rupture of one of these vessels caused a hemorrhage into the vitreous. Kipp[1] and Liebreich[2] rightly maintain that a vein accompanying the hyaloid artery does not exist.

It has been claimed that congenital posterior polar cataract is caused by an improper or incomplete resorption of the lenticular capsule and the remains of the hyaloid system. W. W. Seely[3] reports a case where vision was reduced to $\frac{20}{100}$, and on examination of the eye a dark thread-like body attached to the posterior capsule of the lens was discovered. It performed vermicular movements whenever the eyeball was moved. The papilla was normal in appearance. This seems to be the only case on record where the remains of the hyaloid artery were attached to the posterior surface of the lens. As a rule, these embryonic remains are observed in one eye only. I have found but one case recorded[4] where a distinct vessel having all the appearances of carrying blood was found running from the optic papilla to the posterior pole of the lens in one eye, while in the other the same arrangement existed, but the artery seemed shrivelled. It carried no blood.

I had an opportunity of observing a case of exceeding interest at Professor Seely's clinic, where the patient, a Russian refugee, presented himself for the relief of a granular conjunctivitis of both eyes. The majority of these cases are thoroughly tested as to the refractive condition both with the ophthalmoscope and test lenses. In the examination with the ophthalmoscope I saw a peculiar picture. The fundus with the entrance of the central artery and its distribution was distinctly visible, while one large vessel passed forward through the vitreous to the region of the posterior surface of the lens. The vessel at this point divided regularly, formed quite a network, and finally passed to the ciliary region, just posterior to the iris. Vision was reduced to $\frac{20}{100}$, and was not improved by glasses. The lens was perfectly clear, the pupil movable, but the patient complained of a distinct blurring which he had always

[1] K's Arch. Ophth., vol. iii.
[2] *Trans. Pathol. Society of London.*
[3] *Trans. Amer. Ophthal.* 1882.
[4] Riebau: *Charité Annal.* i., p. 648.

noticed in the right eye as long as he can remember any thing. The left eye was normal in every respect. This case is of special interest, since it presented the vessels almost exactly as they are distributed during the development of the eyeball, yet the capsule and lens were absolutely clear. The vessels were not mere threads or fibres, but presented a distinctly red appearance. The patient, residing only temporarily at Cincinnati, was kept under observation only a few days.

The literature on this subject records but comparatively few cases where the persisting or embryonic vessels in the vitreous continue to carry blood, while but a single case is known to me where the arrangement of the vessels was somewhat similar, viz., that of Hirschberg. In that instance some critics claim the vessels were new formations, instead of embryonic remains, on account of the existence of retinitis in the other eye.

In the author's mind there is little doubt that the criticism in Hirschberg's case is unjust, and was made on account of its exceeding rarity. The impression made on the observer when he first sees such a case is one of doubt until he has studied it carefully; he hesitates in making a diagnosis, as he rarely sees such a picture. It has been claimed by competent writers [1] that the hyaloid artery has an accompanying vein. Upon a careful investigation of the subject and the cases reported, we are firmly led to believe that there is no hyaloid vein, nor does there seem to be any necessity for one. The branches of the artery inosculate with small vessels coming from the ciliary processes and the iris, and in this manner the blood escapes without the intervention of a system of veins, as is the arrangement in all other organs. Vision in these patients as a class is much reduced, and in the majority of cases reported, myopia of a high degree was found.

Those who may have the good fortune to examine such an eyeball *post mortem* as reported by Hirschberg, or as seen by the writer, might contribute some valuable information to the subject of the development of the eye. Aside from the rarity of these cases that would be the only interest connected with them.

[1] *Recueil d'ophthal.*, 1883.

CHOLESTERINE IN THE EYE.

By C. W. TANGEMAN, M.D.,

ASSISTANT TO THE CHAIR OF OPHTHALMOLOGY AT THE MEDICAL COLLEGE OF OHIO.

CASE 1.—James Mc S., a healthy, white laboring man, æt. 48, when making application at my office complained that he was gradually losing his sight in the right eye, while vision of the left eye had been lost by accident some years ago. A careful cross-examination brought out the following history: About three years since the patient received a perforation wound of the eye. The tang of a fork had penetrated the cornea, lacerated the the iris, and wounded the lens. Sight was almost immediately obliterated, although the patient enjoyed good vision previously. The eye became intensely red and caused much pain and suffering. These symptoms passed away in the course of a few months, leaving the globe somewhat smaller and softer than normal. The pupil was smaller than normal, and irregular; while the iris was a little tremulous. The cornea still showed the point of perforation by a small opacity. The aqueous in the anterior chamber had the appearance of being turbid, with a distinct line of deposit, seen in the lower angle of the anterior chamber. By means of the oblique illumination the turbidity was seen to be caused by a mass of silvery-white flocculi, or crystals, in large quantities. When the eye was at rest this mass gravitated to the bottom, and formed the deposit above spoken of, while on the slightest movement a spray of shining crystals floated round and round in the anterior chamber, passing in and out through the pupil. With the ophthalmoscope the beauty of the picture was very much enhanced. The red reflex from the fundus of the eye made an impression on the observer, that is best expressed in the words of Dr. W. W. Seely, nearly a quarter of a century ago,[1]

[1] *Cincinnati Lancet and Observer*, 1866

Reprinted from the ARCHIVES OF OPHTHALMOLOGY, Vol. xvii., No. 4, 1888.

when describing a case of cholesterine in the vitreous humor. Viz.: "It seemed as though one was looking at a miniature sensible horizon, with all the stars shooting chaotically from their places." The spectacle is a grand one, and was repeated as often as the patient moved the eyeball. A few moments, rest, and the vitreous was again sufficiently clear to see the fundus distinctly. By the reflected light the crystals in the vitreous had a yellowish or golden appearance, while in the anterior chamber with the refracted light they were silvery white. The vitreous, which was liquefied, was completely filled with the same mass. The lens had become absorbed, permitting a ready interchange of fluid between the anterior chamber and the vitreous body, which was shown by the manner in which the cholesterine crystals passed from one chamber to another when the eye was rapidly moved. This patient was kept under observation for some time, but the foreign mass, which while not examined microscopically was undoubtedly cholesterine, did not perceptibly diminish or increase. It did not seem to aggravate the symptoms present, or disturb the vision, which was only quantitative. If this mass is being constantly absorbed, it certainly was just as rapidly reproduced, since it appeared to remain the same for over a year, when the patient was finally lost track of.

Case 2.—David S. presented himself at Prof. Seely's clinic, because his eye became periodically inflamed. The patient, a bar-tender, had received a blow on the eye with the fist about two years prior to the application for treatment. From present symptoms, the injury had produced a dislocation of the lens. Intense inflammation of the eye set in, which passed away in the course of about three months without any treatment. The pupil appeared irregular, with a few posterior synechiæ; vision nil. The eye had the appearance of secondary glaucoma. It was subject to occasional attacks of inflammation, which left the eyeball painful for some weeks. The anterior and posterior chambers were filled with a mass of cholesterine crystals, which moved about in the aqueous humor very rapidly upon the slightest movement of the eyeball. A few moments quiet of the eyeball caused a gravitation of the mass to the bottom of the anterior chamber, which gave it much the appearance of hypopyon.

Cases have been recorded where the quantity of cholesterine present in the aqueous humor caused a dimness of

vision when it was agitated a while, but when the eye was kept perfectly quiet for some time the patient could read with the greatest ease.

The first case reported is of interest, since instances are rare where the cholesterine was found present alike in the aqueous chambers and vitreous body. True, these two cases, like the majority reported, had both at some time received an injury to the eye, which has always been looked upon by all writers as the prominent feature in the case, or possibly the causative agent. In a dissertation on "the formation of cholesterine in the human eye" by Schaumberg, we find that the subject formerly was looked upon as a disease called cholestearitis (Desmarres).

That these cases were of frequent occurrence during the time when discision of the lens was practised is readily understood, since the dislocated lens not infrequently set up an inflammation which acted similar to the presence of a foreign body in the eye. The cases described by Desmarres,[1] Stout,[2] and Sichel are all such cases where the operator attempted to remove the cataractous lens from the pupillary area by dislocating it into the vitreous chamber. The presence of cholesterine crystals in the eyes operated on as above stated was not due to the liberation of the cholesterine, always found in the lens as some writers think, but carried there and deposited by some process set up by the foreign body. The statement made by Mc Namara in his late work,[3] that "these cases are quite frequent," must be understood. He has made his observations in India, where the operation for the removal of the opaque lens above mentioned, is so frequently resorted to. One thing seems certain, that we cannot look upon any of these cases as idiopathic, nor as a disease, since the presence of cholesterine in the eye may only mean that certain pathological changes have taken place in some of the tissues. It may be observed in the anterior chamber without an affection of the lens, or in the retina without a

[1] "*Traité théor. et prat. des mal. des yeux*," 1847, p. 665.
[2] *Annal. d'oculistique*, 1846, p. 74; *ibid.* p. 167.
[3] McNamara, "Diseases of the Eye."

liquefaction of the vitreous. Yet it has been observed in the vitreous when no disease of this structure could be diagnosed. Its presence in the vitreous does not cause its liquefaction, nor is it likely to be a result of the liquefaction, but the same cause acting on the vitreous body or other structures of the eye may determine a deposition of this excrementitious principle, which is constantly found in the blood. Theories as to the source of cholesterine crystals are few and very unsatisfactory. It is known that the lens and vitreous humor contain some of this principle physiologically, but not in the crystaline form, nor in quantities such as is often seen in the eye, even though it could all be extracted by some process. Lehmann suggests that it is a product of decomposition, but since we know that it is constantly found in the blood, this theory need receive but little consideration. Some authors attribute this condition to an inflammation of the choroid, iris, or retina, but it has been observed a number of times when the eye was in a perfectly healthy condition. Seely,[1] in an article on cholesterine in the vitreous suggests that its presence in the crystalline form is due to a certain morbid state in which its association with the other normal elements is broken, which causes its precipitation in the form of crystal. For a long time it was held that only diseased eyes contained cholesterine. Wolf[2] associates liquefaction of the vitreous with the presence of cholesterine while the vitreous is not by any means diseased in all of these cases. Physiologists claim that the brain, and possibly to a limited extent the general nervous system, is the source of cholesterine, the blood coming from the brain, containing a larger proportion than the blood flowing toward this organ. In the liver it is again separated from the blood, when it passes into the intestine with the biliary secretion. Just how it is formed in health, where a certain quantity is daily excreted, is not known unless we can accept the theory of Rocafull,[3] namely, that it is due to a chemical decomposition of the albu-

[1] *Lancet and Observer*, 1866.
[2] "Diseases and Injuries of the Eye," p. 250.
[3] *Monatsbl. f. Augenheilkunde.*

minoids. R. reports a case of cholesterine of the lens and attributes its presence to the origin as just mentioned. Experiments show that patients afflicted with cirrhosis of the liver have a larger amount proportionately of this principle in the blood, but only because this disease hinders its proper elimination.

The author is now investigating this class of cases with the ophthalmoscope, with a view to determine whether in these patients where the blood is overcharged with cholesterine it is more likely to be found in the eye. In the four cases reported by Webster Fox,[1] that he calls idiopathic, vision was not materially disturbed, nor was there a lesion of any of the structures of the eye discovered.

Heretofore the presence of cholesterine in the eye and some pathological condition of this organ were always associated. The report of this observation only confirms in the author's mind the belief that cholesterine *may be carried* by the lymph circulation and deposited in the various tissues. It happens occasionally that even rare affections are accidentally associated with well-recognized diseases, which, when considered from an etiological view, is very apt to lead the observer astray. Courtade,[2] reports a case of synchysis scintillans, where these appearances were observed in both eyes two months after the patient had had a shanker, and attributes the presence of cholesterine to syphilis. Possibly the cholesterine disappeared under the administration of iodide of potassium and mercurial ointment. There can be no doubt but that such an association of symptoms was purely accidental. The presence of these crystals in the retina,[3] ciliary body, or anterior chamber,[4] without liquefaction of the vitreous or disease of the retina or lens, would appear to indicate at least that they were not formed there, but carried there by some circulating medium. In the cases termed idiopathic the cause could not be determined by the observer. After analyzing the literature of this subject, we are almost

---

[1] KNAPP'S ARCHIVES, vol. xii., p. 179.
[2] *Union méd.*, xxxix., p. 17.
[3] A. v. Graefe, *Graefe's Archives*, vol. ii., p. 219.
[4] De Wecker, *Graefe und Saemisch.*

forced to the above explanation. A destructive inflammation of any or all the various structures of the eye could not in and of itself produce cholesterine. It may get into the anterior chamber, as is shown by the patient observed by Van Moll,[1] where the anterior chamber was well filled with cholesterine, which rapidly re-appeared after paracentesis of the cornea had been made. The eye in this case eventually became amaurotic, which necessitated enucleation. A microscopic examination of the lens and its capsule showed that these structures had always been free of any diseased process. The source of the cholesterine was due to a detached retina caused by injury, which found its way into the anterior chamber—how? Why these cases are not more frequently met with when the causes to which the affection has been attributed are daily observed, is difficult to explain.

Little or nothing need be said about the *treatment* in these cases; where the eye is not diseased the foreign mass disappears without any damage to the eye, while its presence in a diseased eye does not change the prognosis. General constitutional treatment has been recommended.[2] Hyvernat reports a case where a large mass of cholesterine was found in the healthy vitreous body, while the choroid was diseased. This case may suggest a solution why so often cholesterine is found in eyes where the vitreous is liquid, as a consequence of disease elsewhere. It has been found in the anterior chamber, where it could not have reached this space only by the lymph circulation. It has been found constituting small tumors of the middle ear,[3] without any manifest disease of this organ. Graefe[4] records a case of purulent inflammation of the lachrymal sac where the discharge contained enormous quantities of cholesterine. Tröltsch[5] speaks of its occurrence in a purulent discharge from the middle ear. It has been found in

[1] *Weekblad. van het Nederl. Tijdschr. voor Geweesk*, ii., 595.
[2] *Lyon médical*, xxxiii.
[3] Kuhn, *Arch. f. Ohrenhkunde.*, vol. xxvi.; Schwartz, *Chirurg. Krank des Ohres*, S. 221.
[4] *Arch. f. Ophthal.*, vol. iii., 2, 357.
[5] *Lehrbuch der Ohrenhkunde.*, S. 425.

abnormal quantities in almost every structure of the body. Its presence could not be explained in any manner than that the lymph or blood circulation, or both, was the channel through which the cholesterine crystal found its way into the various structures. From the observations of Seely, Fox, and others, the term scintillating synchysis is objectionable, inasmuch as we may find cholesterine in the vitreous, or even other parts of the eye, without a liquefaction of the vitreous humor.

G. P. PUTNAM'S SONS, PRINTERS
NEW YORK